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Visual motion detection in patients with absent vestibular function

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Abstract

Labyrinthine defective subjects (LDS) experience oscillopsia during head movements due to the absence of the vestibulo-ocular reflex (VOR). The purpose of this study was to compare horizontal and vertical visual motion detection in LDS during (i) body-stationary and (ii) horizontal whole-body oscillation conditions. Twelve LDS and controls detected the onset of drift direction of a grating that moved with accelerating velocity. Thresholds were raised in the patient group in both conditions. The loss of the VOR per se cannot explain raised thresholds in the body-stationary condition nor during whole-body (horizontal) oscillation with vertical grating motion. Findings indicate changes in visual processing that make LDS less sensitive to visual motion. It is postulated that these changes are due to adaptive mechanisms involved to reduce oscillopsia.

Keywords: Visual motion perception; Bilateral vestibular failure; Oscillopsia; Velocity detection

1. Introduction

The function of the vestibulo-ocular reflex (VOR) is to stabilize gaze during head movements. Head movements include rotations (information from the semi-circular canals) and translations (linear displacements; information from the otoliths). The VOR generates slow phase, compensatory eye movements in the opposite direction to head motion at short latencies of approximately 16 ms (Gauthier & Vercher, 1990; Maas, Huebner, Seidman, & Leigh, 1989). Visual blurring and oscillopsia (illusory movement of the environment due to excessive slip of images upon the retina) develops during head movements if the VOR is sufficiently impaired.

Bilateral vestibular loss can be caused by exposure to ototoxic medication (usually gentamicin), bilateral vestibular neuronitis, and meningitis. However, the cause of bilateral vestibular loss is often obscure (idiopathic) (Rinne, Bronstein, Rudge, Gresty, & Luxon, 1998). Its onset may be gradual, patients unconsciously compensating for vestibular loss, in which case oscillopsia is minimal. Even in cases where onset is abrupt, symptoms of oscillopsia on head movement appear to diminish with time, as a result of a number of compensatory processes developing over time (Bhansali, Stockwell, & Bojrab, 1993; Bronstein, Morland, Ruddock, & Gresty, 1995; Maw, 1971). It was this aspect of recovery that prompted our study.

We developed a motion detection task to measure change in tolerance to retinal slip in patients with involuntary eye movements who did not suffer from oscillopsia (congenital nystagmus). In these patients, we found raised visual motion detection thresholds when tested with the head stationary (Shallo-Hoffmann, Bronstein, Acheson, Morland, & Gresty, 1998). When oscillopsia does occur, both in patients with ocular oscillations and ocular-motor palsies, studies have shown that the amplitude of the oscillopsia is smaller than the amplitude of retinal slip (Büchele, Brandt, & Degner, 1983; Wist, Brandt, & Krafczyk, 1983). Both sets of findings indicate that oscillopsia can be partially suppressed. In such cases, retinal slip is less likely to be perceived, therefore, oscillopsia is not appreciated. The purpose of the present study was to test the hypothesis that a contribution to the abatement of oscillopsia in patients with bilateral vestibular loss is due to a reduction in sensitivity to slow visual motion. We applied the visual motion detection task both with the head stationary and during whole-body oscillation to investigate if any change in motion detection occurred as a function of the test condition.

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2. Materials and methods

2.1. Subjects

Twelve patients (34–74 years; mean: 52.5 years) and twelve age-matched control subjects (33–73 years; mean: 52.4 years) performed a motion detection task. The patients, recruited from the out-patient neuro-otology clinics in London, suffered from various degrees of unsteadiness and oscillopsia on head movement due to bilateral peripheral vestibular loss. Absence of VOR was confirmed by yaw velocity step rotation in the dark (±80 deg/s) and bithermal caloric irrigation, both with optic fixation and Frenzel's glasses. Patients underwent complete neuro-ophthalmological and -otological examination and none of the patients had spontaneous or gaze evoked nystagmus.

Control subjects were healthy age-matched staff or relatives of the patients who did not have a history of hearing loss or vestibular symptoms (Table 1). It was the first time that any of the subjects (patients and controls) participated in motion perception tasks and they were naive to the purposes of the study. Investigations were performed according to the guidelines of the Declaration of Helsinki and were approved by the hospital's medical ethics committee. Subjects were fully informed about the nature of the procedures and gave their written consent before beginning the experiment.

2.2. Visual motion detection tasks

2.2.1. Stimulus

A detailed account of the apparatus used to determine threshold values, in a similar paradigm, has been described elsewhere (Shallo-Hoffmann et al., 1997). In brief, the stimulus consisted of a back-projected, grey scale, sinusoidal grating (0.23 cycle/deg, 37 cm in diameter), subtending 18.5 deg. The grating (either a vertical grating moving horizontally or a horizontal grating moving vertically) was vignetted with a circular mask, to eliminate both flicker and motion cues by shading off and diffusing the edge of the stripe into the background.

2.2.2. Static condition

Dark-adapted subjects sat in a chair, 130 cm before a screen (236 cm × 145 cm), and binocularly viewed the stimulus described above with their head and chin restrained by rests.

2.2.3. Whole-body oscillation condition

Subjects binocularly viewed the stimulus described above while passive horizontal whole-body oscillation was performed at 1.0 Hz with a peak head velocity of 34 deg/s. Seat belts, foot, leg, head and chin rests were used to restrain body motion.

2.2.4. Determination of contrast threshold values for visibility of a static stimulus

Before the motion detection task was performed in either test condition, the contrast of a static grating was adjusted to the point of subjective visibility for each subject by a staircase procedure, in which 1 of 40 contrast levels was presented on every trial. Estimation of contrast threshold always started with contrast below threshold for detecting the grating. Since subjects were dark adapted, this procedure of starting with an ascending staircase insured that subject's maintained dark adaptation to minimum ambient luminance of the detected grating. The range of contrast values was selected such that the midpoint was approximately at contrast threshold detection level, that is, the least contrast re-

Table 1
Clinical status of 12 patients with complete vestibular loss and 12 age-matched controls

Patients code	Age/sex	Bilateral vestibular loss	Bithermal calorics	Post-rotary nystagmus (80 deg/s)	Duration of loss before testing	Control subject code–Age/sex
1P	54/F	Idiopathic	Absent	None	4 years	5C–48/M
2P	47/F	Idiopathic		None	12 years	2C–47/M
3P	54/M	Post-bacterial meningitis	Absent	None	10 years	4C–50/M
4P	68/F	From gentamicin	Severely reduced	NA	1 year	1C–71/F
5P	51/M	Idiopathic		None	3 year	7C–51/M
6P	34/M	Idiopathic	Absent	None	3 year	10C–33/M
7P	55/M	Idiopathic	Absent	None	2 weeks	11C–57/M
8P	64/M	Idiopathic	Absent	None	11 years	8C–60/M
9P	74/M	Idiopathic		None	7 years	3C–73/M
10P	36/M	Neurosarcoidosis	Absent	None	4 years	6C–36/M
11P	43/M	Idiopathic	Absent	None	4 years	9C–48/F
12P	50/F	Bilateral vestibular neuronitis		None	9 months	12C–48/M
Mean age	52.5 yrs/8M;4F	M = male; F = female	yrs = years	NA = not available		51.8 yrs/10M;2F

quired for detection of a static grating 50% of the time. Contrast was then set just above this threshold value. The specific increment above contrast was chosen to ensure low but reliable visibility, although the increment was never less than 0.5% nor greater than 0.9%. Setting contrast just above detection threshold was a control to ensure that all participants were tested at the same level of detection difficulty for their specific visual acuity.

2.2.5. Determination of vertical and horizontal motion detection

Motion detection thresholds were measured under two conditions (static and whole-body motion) in a (a) *vertical detection task* (when a horizontal grating moved either upward or downward) and a (b) *horizontal detection task* (when a vertical grating moved either rightward or leftward). The tasks were performed in separate sessions, either with an hour pause between test sessions or on separate days. A motion detection task followed directly after the contrast threshold value of the stimulus was ascertained for each individual, using the procedure described above.

The stimulus grating was initially stationary, and started to accelerate at a constant rate of 0.09 deg/s^2. A small constant acceleration was used to provide a sensitive, meaningful measurement of motion detection. The instruction to the subject was to report the drift direction of the grating as soon as the grating first appeared to move. There were at least 12 trials in each condition, randomised for 6 presentations in each direction. That is, each subject performed a vertical and horizontal motion detection task under both static and whole-body oscillation conditions. The order of presentation of the test condition (static or whole-body oscillation) and task (horizontal or vertical motion) was balanced. The order of presentation of the stimulus direction (right or left for horizontal motion; up or down for vertical motion) was randomised. A testing session for one condition lasted approximately 45 min.

3. Results

Velocity thresholds were raised in the patient group in all test conditions as compared to the age-matched control group (Tables 2 and 3, nested ANOVA, $p < 0.0001$). Variations were found within each group of subjects, that is, some individuals had slightly higher thresholds than others within each test group (nested ANOVA, $p < 0.0001$ for all test conditions). No differences were found due to direction of the motion except in the static, vertical motion condition in the patient group (nested MANOVA, $p < 0.0001$). The mean value for upward motion was 0.46 deg/s as compared to downward motion (0.36 deg/s).

Unlike the control group, mean vertical motion detection thresholds increased under whole-body oscillation in the patient group (vertical up: $t = -2.5$, $p < 0.025$; vertical down: $t = -2.53$, $p < 0.01$; Table 3).

Subjects did not report a sense of vection when performing the static detection task. Three patients (1P, 4P and 10P) could not reliably perform the horizontal detection task during whole-body motion (Table 2). Although they always reported that they could see the grating, they could not reliably detect the direction of the grating motion. These three patients were able to perform the vertical motion detection task (Table 3).

Control subjects' threshold values did not change during the whole-body oscillation condition as compared to the head-static condition (horizontal motion detection: $t = 0.5128$, df-22, ns; vertical motion detection task: $t = 0.116$, df-22, ns).

4. Discussion

The main finding in this study is that motion detection scores were raised in the patient group in all conditions; head-static and whole-body oscillation when motion was both in the horizontal and vertical direction. Horizontal motion detection thresholds during horizontal whole-body oscillation can be expected to be abnormal as a direct consequence of deficient compensatory vestibular eye movements, that is, gaze instability under the body oscillation condition. Indeed, three of the 12 patients could not reliably perform the task. However, raised thresholds during the head stationary conditions, and in the *vertical* motion detection task during *horizontal* whole-body oscillation, lends evidence that adaptive mechanisms contribute to the change in motion detection since all patients could reliably perform these tasks.

In many instances total or subtotal loss of the VOR is a clinically catastrophic event. Patients with vestibular failure due to acute conditions such as meningitis or ototoxicity initially experience intense, disabling oscillopsia. Despite the irreversible nature of the vestibular lesion, most patients improve symptomatically and animal as well as clinical work established that the enhancement of neck-ocular reflexes (Bronstein & Hood, 1986; Dichgans, Bizzi, Morasso, & Tagliasco, 1973; Kasai & Zee, 1978) is an important compensatory mechanism mediating such recovery. However, the degree of potentiation of the cervico-ocular reflex after the vestibular injury is not correlated with the degree of subjective improvement of oscillopsia and, therefore, visual–perceptual mechanisms have been suspected to participate in the recovery process (Bronstein & Hood, 1987). Our current finding of raised thresholds under static conditions, and the further rise by body oscillation, lends support to the view that patients with

Table 2
Mean horizontal motion detection scores (deg/s ± standard deviation); Static-head and whole-body oscillation conditions

Code patient	Static head		Whole body	
	Horizontal		Horizontal/dynamic (34 deg/s)	
	Right	Left	Right	Left
1P	0.31 ± 0.13	0.55 ± 0.16	Failed	Failed
2P	0.60 ± 0.12	0.58 ± 0.09	0.90 ± 0.23	0.82 ± 0.21
3P	0.75 ± 0.14	0.59 ± 0.13	1.74 ± 0.26	1.46 ± 0.31
4P	0.59 ± 0.09	0.59 ± 0.13	Failed	Failed
5P	0.39 ± 0.14	0.38 ± 0.12	0.54 ± 0.24	0.67 ± 0.21
6P	0.43 ± 0.08	0.38 ± 0.03	0.67 ± 0.15	0.46 ± 0.11
7P	0.48 ± 0.09	0.30 ± 0.08	0.81 ± 0.42	0.87 ± 0.36
8P	0.37 ± 0.10	0.33 ± 0.06	1.02 ± 0.14	1.03 ± 0.13
9P	0.39 ± 0.07	0.39 ± 0.09	0.47 ± 0.09	0.56 ± 0.11
10P	0.50 ± 0.09	0.50 ± 0.09	Failed	Failed
11P	0.57 ± 0.09	0.75 ± 0.14	1.22 ± 0.36	1.19 ± 0.16
12P	0.39 ± 0.05	0.64 ± 0.18	0.70 ± 0.16	0.80 ± 0.14
Mean	0.48 ± 0.13	0.50 ± 0.14	0.90 ± 0.39	0.87 ± 0.31
Control				
1C	0.20 ± 0.07	0.23 ± 0.09	0.25 ± 0.07	0.27 ± 0.08
2C	0.37 ± 0.06	0.42 ± 0.12	0.47 ± 0.12	0.31 ± 0.05
3C	0.26 ± 0.08	0.33 ± 0.09	0.22 ± 0.09	0.29 ± 0.10
4C	0.42 ± 0.04	0.39 ± 0.06	0.40 ± 0.14	0.36 ± 0.10
5C	0.42 ± 0.04	0.28 ± 0.08	0.44 ± 0.07	0.49 ± 0.15
6C	0.21 ± 0.05	0.27 ± 0.08	0.32 ± 0.04	0.32 ± 0.05
7C	0.27 ± 0.08	0.24 ± 0.08	0.33 ± 0.08	0.34 ± 0.04
8C	0.25 ± 0.08	0.29 ± 0.10	0.26 ± 0.05	0.25 ± 0.01
9C	0.30 ± 0.06	0.29 ± 0.10	0.34 ± 0.09	0.29 ± 0.10
10C	0.21 ± 0.08	0.27 ± 0.12	0.20 ± 0.34	0.23 ± 0.05
11C	0.21 ± 0.07	0.22 ± 0.07	0.32 ± 0.09	0.23 ± 0.09
12C	0.34 ± 0.04	0.34 ± 0.05	0.34 ± 0.02	0.30 ± 0.04
Mean	0.29 ± 0.01	0.30 ± 0.06	0.32 ± 0.08	0.31 ± 0.07
ANOVA	$F = 19.800$ (23,134)	$p < 0.0001$ right	$F = 31.836$ (20,117)	$p < 0.0001$ right
	$F = 13.556$ (23,136)	$p < 0.0001$ left	$F = 35.589$ (20,114)	$p < 0.0001$ left

bilateral vestibular loss ignore slow motion to avoid oscillopsia. It is an adaptive mechanism to aid perceptual–visual stability in the presence of the excessive retinal image slip encountered during head movements. Motion detection evaluations may become a useful tool to estimate if patients are able to use this strategy to minimize symptoms. This notion has support from a study by Grunfeld, Morland, Bronstein, and Gresty (2000), in which eye movement recordings in avestibular patients measured the amount of retinal image slippage during whole-body oscillation. Grunfeld et al. (2000) found that retinal image speed was, contrary to their expectations, inversely related to the clinical disability brought about by the oscillopsia. This ocular-motor finding agrees with our current perceptual results; the implication is that patients adapt to the oscillopsia due to loss of the VOR by raising visual motion perceptual thresholds that, in turn, leads to an increased tolerance to retinal image slip.

Morland, Bronstein, and Ruddock, 1995, and Grünbauer, Dieterich, and Brandt, 1998, are the only other studies, as far as we know, that have investigated motion perception in patients with bilateral vestibular loss. Both studies yielded mixed results. Morland et al. (1995) measured visual velocity discrimination in four patients. One of the four patients demonstrated differences in velocity discrimination under a whole-body oscillation condition and the authors suggested that the finding was due to central suppression of motion perception to reduce oscillopsia. Grünbauer et al. (1998) measured the latency it took a subject to respond to a single suprathreshold light spot (diameter 1 deg of visual angle) that moved at a constant velocity of 40 arcmin/s. Latency to report the movement of the spot in the horizontal direction was significantly longer for four of the eight patients with either subtotal or complete vestibular loss as compared to age-matched control subjects. The authors interpreted these findings as evidence for impaired motion perception caused by a central visual mechanism that suppresses oscillopsia. Although the interpretation from both studies is compatible with the findings presented here, the evidence was less compelling since only one of four patients in the former study showed differences in velocity discrimination and only half the

Table 3
Mean vertical motion detection scores (deg/s ± standard deviation); Static-head and whole-body oscillation conditions

Code patient	Static head		Whole body	
	Vertical		Vertical/dynamic (34 deg/s)	
	Up	Down	Up	Down
1P	0.58 ± 0.09	0.28 ± 0.12	0.54 ± 0.14	0.84 ± 0.25
2P	0.22 ± 0.05	0.35 ± 0.09	0.63 ± 0.18	0.48 ± 0.22
3P	0.58 ± 0.20	0.70 ± 0.14	0.85 ± 0.16	0.77 ± 0.21
4P	0.37 ± 0.17	0.58 ± 0.08	0.81 ± 0.21	0.61 ± 0.15
5P	0.49 ± 0.21	0.32 ± 0.15	0.53 ± 0.18	0.29 ± 0.04
6P	0.28 ± 0.01	0.36 ± 0.05	0.42 ± 0.08	0.43 ± 0.08
7P	0.42 ± 0.10	0.30 ± 0.07	0.65 ± 0.14	0.46 ± 0.20
8P	0.64 ± 0.16	0.22 ± 0.05	0.67 ± 0.17	0.36 ± 0.05
9P	0.32 ± 0.12	0.27 ± 0.17	0.36 ± 0.05	0.40 ± 0.16
10P	0.42 ± 0.09	0.39 ± 0.05	0.41 ± 0.19	0.40 ± 0.08
11P	0.60 ± 0.20	0.31 ± 0.10	0.81 ± 0.16	0.69 ± 0.20
12P	0.54 ± 0.07	0.31 ± 0.06	0.63 ± 0.29	0.77 ± 0.29
Mean	0.46 ± 0.14	0.36 ± 0.14	0.61 ± 0.16	0.54 ± 0.19
Control				
1C	0.24 ± 0.07	0.24 ± 0.06	0.24 ± 0.04	0.23 ± 0.05
2C	0.39 ± 0.05	0.30 ± 0.06	0.34 ± 0.05	0.30 ± 0.05
3C	0.32 ± 0.17	0.30 ± 0.17	0.20 ± 0.07	0.18 ± 0.05
4C	0.36 ± 0.03	0.39 ± 0.06	0.27 ± 0.09	0.30 ± 0.11
5C	0.31 ± 0.07	0.36 ± 0.10	0.39 ± 0.12	0.41 ± 0.11
6C	0.26 ± 0.08	0.20 ± 0.03	0.25 ± 0.04	0.23 ± 0.04
7C	0.26 ± 0.04	0.32 ± 0.04	0.31 ± 0.07	0.34 ± 0.11
8C	0.22 ± 0.03	0.25 ± 0.07	0.21 ± 0.05	0.28 ± 0.10
9C	0.20 ± 0.04	0.35 ± 0.06	0.39 ± 0.05	0.28 ± 0.05
10C	0.19 ± 0.05	0.14 ± 0.05	0.19 ± 0.03	0.15 ± 0.06
11C	0.31 ± 0.10	0.22 ± 0.07	0.47 ± 0.12	0.22 ± 0.07
12C	0.33 ± 0.08	0.27 ± 0.03	0.32 ± 0.11	0.34 ± 0.10
Mean	0.28 ± 0.07	0.28 ± 0.073	0.30 ± 0.09	0.27 ± 0.07
ANOVA	$F = 8.642$ (23,117)	$p < 0.0001$ up	$F = 15.274$ (23,122)	$p < 0.0001$ up
	$F = 9.490$ (23,116)	$p < 0.0001$ down	$F = 10.614$ (23,122)	$p < 0.0001$ down

patients had longer latency responses in the latter study. Findings were in complete accord in our study. All 12 patients with bilateral vestibular loss showed evidence of raised thresholds in motion detection. We suggest that two factors may have affected the mixed findings reported by Morland et al. (1995) and Grünbauer et al. (1998): (1) Some of the patients may have had some residual vestibular function. In our study all patients had an absent or a severely reduced response on bitermal caloric testing and an absent nystagmus response with rotary chair testing. (2) A significant factor that may have affected findings in the above mentioned studies may have involved the use of suprathreshold tasks. Suprathreshold tasks are less sensitive than threshold tasks. All subjects (patients and controls) must be put under an *equal task demand, an equal level of discrimination or detection difficulty*, before performing the critical measurement. For example, in our study, all subjects were held under the same task demand by controlling, at threshold, the visibility of the stimulus for each individual. Without controlling for this criteria, between and within subject variability can either camouflage or exaggerate findings.

We and others have shown that patients with congenital ocular oscillations (Abadi, Whittle, & Worfolk, 1999; Bedell, 1992; Bedell & Bollenbacher, 1996; Dieterich & Brandt, 1987; Shallo-Hoffmann et al., 1998) and, in this study, patients with bilateral vestibular loss have raised thresholds for motion perception. We do not interpret these findings as "impairment" of motion perception in either patient group. Raised thresholds in motion perception reflect a perceptual-adaptive change in the processing of sensory visual motion. It is useful to ignore slow motion to counter-excessive retinal slip and aid in avoiding oscillopsia. The defect or impairment is the excessive retinal slip found in congenital nystagmus or caused by a malfunctioning VOR during head motion rather than the patients' processing of motion perception.

5. Conclusions

Raised thresholds in motion detection observed in the absence of whole-body oscillation, and during whole-body oscillation with vertical grating motion, cannot be

explained by a defective VOR and lend evidence that perceptual-adaptive, compensatory mechanisms are involved to reduce oscillopsia.

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